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REPORT

CD NO.

50X1-HUM

COUNTRY	USSR	DATE OF INFORMATION	1947
SUBJECT	Medical - Toxicology		
HOW PUBLISHED	Bimonthly periodical	DATE DIST.	9 Aug 1949
WHERE PUBLISHED	Moscow	NO. OF PAGES	3
DATE PUBLISHED	May - Jun 1947		
LANGUAGE	Russian	SUPPLEMENT TO REPORT NO.	

THIS IS UNEVALUATED INFORMATION

SOURCE Farmakologiya i Toksikologiya, Vol X, No 3, 1947, (FDB Per Abs 14125)

ANTIFREEZE POISONING DURING WORLD WAR II

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Seventeen cases of antifreeze poisoning were examined at the Kuybyshev medico-judicial post-mortem examination room. All were due to accidental intake of antifreeze instead of alcohol. The type and result in respect to these cases can be divided into two groups according to the chemical composition of the antifreeze consumed.

Antifreeze preparations are divided into three groups according to their chemical composition: (1) mixtures of methyl alcohol and glycerine, (2) mixtures of ethyl alcohol and glycerine, and (3) ethylene glycol with or without glycerine.

Ethylene glycol is gradually replacing other antifreeze preparations. It is called prestone in America and glisantin in Germany. It is a yellowish, rather viscous liquid with a sweetish taste and no smell. Its specific gravity at 20 degrees is 1.05 - 1.08, freezing point -40 degrees, and boiling point 197 degrees. Chemically, it is a secondary alcohol $CH_2OH - CH_2OH$.

Three stages can be distinguished in the clinical picture of ethylene glycol poisoning:

1. Refractory, characterized by signs of mild drunkenness appearing immediately or soon after the intake of antifreeze.

2. Cerebral, characterized by the change in the narcotic effect on the ethylene glycol to comatosis; the patient usually expires on the second day.

3. Nephritic, when the patient begins to develop signs of anuria and expires on the 13th or 14th day, showing signs of azotemic uremia.

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In the case of patients who have died in the first 2 days (in the comatose condition) a considerable accumulation of blood in the cerebral matter is noticeable. Microscopically, and occasionally even macroscopically, minute hemorrhagic effusions accompanied by encephalic degeneration are observed in various parts of the brain. Localized hemorrhagic effusions are frequently observed on the posterior surface of the ventricles of the heart. The urine specimen indicates a fairly large amount of white precipitate of calcium oxalate.

In the case of patients who have died in the nephritic stage on the 12th - 14th day, the kidneys are enlarged and adhesion of the capsules and the parenchyma is observed in places where there are hemorrhagic effusions. Cyanosed areas with vacuolated tissues are observed in the center of these hemorrhagic effusions. Maximum vacuolation is observed in the epithelium of the winding ducts and Henle's loops and this condition is accompanied by some cellular degeneration. Strangely-shaped calcium oxalate crystals are found in the lumens of the ducts. Hydropic degeneration of liver cells is also observed.

These cases of poisoning were caused by two different antifreeze preparations. Eight of these were intoxicated by alcohol-glycerine mixtures. We shall not dwell on this first group, since the clinical and post-mortem examinations resembled those in alcohol poisoning and diagnosis is fairly easy.

The second group included nine cases. In these, poisoning was due to antifreeze preparations whose basic constituent was ethylene glycol.

All these cases of ethylene glycol poisoning were acute and fatal. Death ensued during the first 24 hours after the intake of antifreeze in seven cases and on the second day in two cases. All the patients were men of from 18 to 42 years of age. They were admitted to therapeutic institutions a few hours after taking the antifreeze, in a critical comatose condition, and the persons accompanying them were generally unable to give a clear account of what had happened. This was the first time that such poisoning had been encountered in Kuybyshev.

It was noted in the case histories that these patients were unconscious; there was no alcohol smell, with only occasional vomiting. The pupils were dilated and reacted feeble to light. The pulse was barely perceptible. Cyanosis of the face and the visible mucous membranes was evident. Stertorous respiration and sometimes foamy expectoration were observed. Periodical clonic spasms, involuntary defecation and urination were also evident.

Laboratory analysis of three in this group was carried out. An increase in the leucocyte count up to 23,100 was recorded. Urinalysis indicated the albumin up to 0.066 %; leucocyte count 5 - 12 per microscopic field and isolated hyaline casts.

Physical examination revealed cyanosis of the skin, especially about the face and neck, and also a pronounced congestion of the conjunctiva. Internal examination showed general signs of asphyxia: plethora of the internal organs, especially the brain and localized hemorrhagic effusions in the lungs and heart, mainly on the posterior surfaces. Hypertrophia and hyperemia of the mucous membrane of the stomach and the intestine with sporadic minute hemorrhagic effusions, i. e., such a macroscopic picture resembles that of methanol poisoning.

A chemical analysis of the internal organs was made to clarify the nature of the poison and ascertained the presence of ethylene glycol.

Thus, in summing up, we can say that poisoning with brain symptoms predominant, resulted in death. Due to this reason, degenerations peculiar to the third stage and characterized chiefly by renal degeneration which can be easily diagnosed by post-mortem examination were not observed in these cases.

We based our diagnosis on the post-mortem examination (which is analogous to methyl alcohol poisoning) and the results from chemical analysis of the internal organs. It should be noted that ethylene glycol usually produces reactions of primary alcoholic intoxication; therefore, cases indicative of positive intoxication must be specially examined for ethylene glycol.

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In these cases, poisoning by ethylene glycol was confirmed chemically in five cases. In two cases the test for ethylene glycol was negative. This is clearly explained by the fact that by the time we examined these cases there was no free ethylene glycol in the organism. Similar condition was observed in those cases where death ensued on the second day after the poisoning. In two cases tests for alcohol produced positive results, but the reaction for ethylene glycol was negative.

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